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SYMPHYSIOTOMY: A RE-APPRAISAL

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This is an account of a personal re-discovery of symphysiotomy, and more especially a plea for wider instruction in the role of symphysiotomy and its *limitations* in obstetrics, in order to help others.

The teaching in some medical schools recently was that 'symphysiotomy is a procedure only mentioned to be condemned'. Pubiotomy was referred to as preferable, but needed special instruments. About both, warnings were uttered about chronic sacro-iliac strain and waddling gait as sequelae.

It is necessary first to describe the circumstances in which a district medical officer may find himself in an African territory. It is in this context that the theme is tendered.

The district in question is several hundred square miles in extent; ranging from one to several thousand feet above sea level. The population is estimated to be several score thousand Africans and a negligible percentage of Whites. The nearest proper Government hospital is several score miles away over gravel roads which are often impassable in the rainy season. There are two hospitals: one, a mission hospital having its own doctor, the nearest colleague, is nearly 30 miles away. There is a daily bus service to all main villages, and a rural 'party-line' telephone system.

The local hospital of 50 beds has the part-time services of the district medical officer for several hours daily. It is staffed by a few White and African State-registered nurses, (who are also registered midwives), assistant nurses, and student nurses. There is an ambulance and a theatre equipped to cope with most emergencies, from amputations to a ruptured ectopic pregnancy.

In the absence of either an assistant or anaesthetist, extensive use is made of local and regional anaesthesia. Major operations are therefore only performed in an emergency.

At the local hospital the medical officer sees only those obstetric patients beyond the scope of the midwives. These are nearly all patients with failed trials of labour brought in late by ambulance from some witch-doctor in the hills.

During the last 2 years, during which period the symphysiotomies were done, there was a total of 550 deliveries including 6 caesarean sections, 12 forceps deliveries, and 17 symphysiotomies.

The first symphysiotomy was performed on a patient who had been in labour for 10 hours in the second stage when I was called to see her. The head had been visible at the vulva for 8 hours. There was foetal and maternal distress. The head was so tightly impacted that forceps could not be applied to the narrow outlet. The patient was febrile. With some hesitation arising from knowledge of the condemnation of the procedure, a symphysiotomy seemed the only possible course to adopt. With great caution I started to translate the word 'symphysiotomy', my only knowledge of the procedure, into action. Under infiltration with a little local anaesthetic the symphysis was literally cut, using what appeared to be the safest instrument, a solid-bladed scalpel. The incision was ¾ inch long, vertically downwards from just below the upper border of the symphysis. Progress was palpated with the left little finger in the wound from time to time. The patient had been placed in the lithotomy position with the stirrup supports turned inwards to prevent too sudden or too wide a separation, which I feared might affect the sacro-iliac joints. (At that stage I was not aware of the much more real and immediate danger to the bladder and urethra.) When sufficient had been cut, the symphysis was gently levered open to 2 cm.

It then opened to 4 cm. on its own when the inert uterus immediately resumed regular contractions to deliver the living child, without forceps.

It appeared to be too easy and simple a procedure. The contraindications and dangers were learnt later. The standard textbooks on midwifery in my possession were not very helpful, nor were my colleagues, whose telephoned advice was unenthusiastic.

The accompanying Table (Table I) calls for special comment upon a few of the cases, and general remarks follow where appropriate.

Case 3

This patient with brim disproportion needed forceps to effect delivery of the 9-lb. baby. In the process the symphyseal gap widened suddenly beyond the safe 3 - 4 cm. to 6.5 cm., resulting in a rending of the arcuate ligaments and a lateral tear of the bladder. Immediate repair was done and an indwelling catheter inserted for 14 days.

Pubic mobility necessitated belt-strapping of the pelvis. A padded leather strap was effective. The patient was discharged well, after a month, and had no complaints on follow-up 6 months and a year later.

After this the next few patients were strapped and confined to bed for 3 weeks, but this was ignored as unnecessary by those patients whose gap had opened to 4 cm. or less. They had loosened their straps and were up within a week without any ill-effects.

On all but a few, a late follow-up was done at 6-monthly intervals by using the good offices of the authority of the Assistant Commissioner to persuade the patients to attend for examination. Where necessary their fares were paid. They were examined for backache, stress incontinence, and their ability to walk, run, and jump. The symphysis was palpated and the degree of movement was estimated clinically by observing them

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TABLE I. SEVENTEEN SYMPHYSIOTOMIES, ALL PERFORMED IN FAILED TRIALS OF LABOUR. (CLINICAL PELVIMETRY IN CM.)

Case	*Inter-cristal*	*Inter-spinous*	*Ex. conj.*	*True conj.*	*Outlet*	*Birth weight lb.*	*oz.*	*Indication*	*Width symphyseal gap opened in cm.*	*Complications*	*End result*
1	23	19·5	18·5	9·5	Small	7	12	Outlet delay	4	Nil	Good
2	24·5	21·5	19	10	Average	6	8	P. occipito-post.	2·5	Nil	Good
3	25	21	17·5	+ 7·5	Small	9	—	High head. Brim dispro-portion	6·5	Vestibular tear and torn bladder satis-factorily repaired	Good
4	24·5	21·5	18	9	Average	6 5	— 8	Mat. distress. Twins dead, undelivered	2·5	Twins died owing to prolapsed cords	Mother fine
5	24	23·5	17·5	+ 7·5	Small	7	8	Brim and outlet delay	4	Nil	Good
6	23·5	21	17·5	+ 7·5	Small	7	—	Brim and outlet delay	4	Nil	Good
7	23·5	21·5	19	10	Small	6	12	Outlet delay	3	Nil	Good
8	22	21	20	11	Small	6	12	Distress and outlet delay	2·5	Nil	Good
9	23·5	19	17·5	+ 7·5	Average	6	12	High head	4·5	Nil	Good
10	24·5	23·5	19	10	Small	6	8	High head and outlet delay	6·5	Vestibular tear, gross haemorrhage	Good
11	23·5	22	17·5	+ 7·5	Very small	7	—	Extreme eclampsia	6·5	Temporary unstable pubic symphysis	Good
12	24	21·5	18·5	9·5	Small	6	12	P. occipito-post.	4	Nil	Good
13	24	22	17·5	+ 7·5	Very small	6	8	High head	7·5	Vestibular tear and tear of bladder. Unstable pubic symphysis	Vesico-vaginal fistula
14	23·5	21·5	17·5	+ 7·5	Small	7	—	Outlet delay	4	Nil	Good
15	25·5	23	19	10	Average	10	12	Previous caesarean	5·5	Nil	Good
16	25	23·5	20·5	11·5	Average	6	8	Breech	2·5	Nil	Good
17	24	22	17·5	+ 7·5	Small	10	—	P. occipito-post.	4·5	Nil	Good

whilst climbing a few steps. No differences were found that were not present in a similar group of normal mothers also examined.

Case 10

This patient had only 1 cm. difference between her intercristal and interspinous diameters. She also had a small outlet. There was gross maternal distress after many hours of delay in the second stage, and the foetal heart was *not* heard; forceps were therefore also used. Unfortunately the arcuate ligaments tore with the widening of the gap, resulting in gross haemorrhage from a vestibular tear. However, the baby *was alive*, and both mother and child did very well.

Case 11

This eclamptic patient was brought in by ambulance from about 30 miles away. She was unconscious and had fits every 2 minutes. The blood pressure was 185/140 mm. Hg between the fits. A catheter specimen of urine was loaded with albumin. There was no consent for operation and it would have taken another hour to prepare for a caesarean section. She was a small woman, only 4 ft. 8 inches tall. A symphysiotomy permitted the foetal head to engage and live delivery was effected by forceps within minutes of her arrival. In spite of adequate strapping it was 6 weeks before her pelvis was stable enough for her to walk normally. The hypertension and albuminuria became normal within 2 weeks of delivery on appropriate treatment. Late follow-up was normal. This patient was simply lucky to escape serious trauma and in retrospect should have had a caesarean section.

Case 13

This is the only really unfortunate maternal case of the series. The patient went into trial labour in hospital. Her

small outlet, especially, was known beforehand. The head remained high after prolonged labour, but consent for interference was withheld until there was maternal distress. A symphysiotomy was then done and the gap opened at first to about 3 cm. and spontaneous delivery was expected. However, there was no progress for hours when increasing distress necessitated further action. Fundal pressure advanced the head sufficiently for mid-forceps, which were used gently, but during a satisfactory advance, a sudden contraction widened the gap to over 7·5 cm. with a tear of the ligaments extending along the right antero-lateral aspect of the bladder for about 2·5 cm. The baby was fine. Immediate repair was done and an indwelling catheter inserted. After a few days the catheter came adrift, but I was not available to replace it until the bladder had become distended and the sutures had given way. An attempt at further repair failed on account of friable tissue and a low-grade infection, despite antibiotics.

There was great mobility of the pubes, but this slowly returned to normal after about 3 months. The vesico-vaginal fistula was closed later by a urological and gynaecological team at a larger centre.

It was only at this stage that I was able to obtain copies of Greig's[1] splendid review of the subject, and Zarate's[2] booklet.

It appears that I had empirically arrived at the same degree of cut that Zarate advises, by using the method described above, dividing the cartilage but leaving most extra-capsular structures intact. In essence Greig effected much the same, but he employed a rather more elaborate surgical approach and also usually used spinal anaesthesia in his own series. Being alone I purposely tried to avoid spinal anaesthesia and caesarean section wherever possible. However, whenever caesarean section became absolutely necessary the patients were given spinal anaesthesia, except for 2 or 3 very shocked patients who had sedation and local infiltration only.

The double fall in blood pressure when spinal anaesthesia is used in obstetrics is too well-founded to be ignored, even when used with prophylactic hypertensives.

The last 4 patients had the benefit of my 'reading-up' the subject, but it was not until treating cases 16 and 17 that I had the courage to use the 'blind' method of Zarate — the 'subcutaneous partial symphysiotomy'. I can, however, recommend it as entirely satisfactory, and the simplest of all techniques.

DISCUSSION

From this small series it has become evident that symphysiotomy is a procedure in its own right with its own special indications and limitations. It appears to be a substitute for neither caesarean section nor forceps, and it seems in retrospect that a case was badly selected if forceps became necessary. It is certainly not easy to select the ideal case. Symphysiotomy should be contraindicated when more than very minor degrees of brim disproportion are present.

It is an ideal procedure in cases of small outlet in an otherwise fair pelvis, and in cases of malrotation (especially failed manual rotation).

There is no doubt that the procedure can be life-saving, and in the above series the only mortality was the loss of twins caused by prolapsed cords unrelated to the symphysiotomy procedure. From the Table it will be noticed that the symphyseal gap opened too widely chiefly in those cases where the true conjugate was less than normal; hence I would suggest an absolute lower limit of, say, 8·5 - 9 cm. for the true conjugate.

SUMMARY

The necessity for symphysiotomy in remote rural obstetric practice is evaluated as an essential and sometimes life-saving procedure.

A small series of 17 cases is reviewed with special comment upon a few of them.

Certain conclusions are drawn and set out above, especially with reference to the lower limit of the true conjugate for the procedure.

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POST-TRAUMATIC ILIACUS ABSCESS*

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An abscess in, or deep to, the iliacus muscle is apparently uncommon, but may occur more often than is believed, because the diagnosis is overlooked in an appreciable number of cases.

Five cases have come to my attention in the course of the past 6 years and, since the diagnosis and treatment are beset by problems and difficulties, this experience seems to be worthy of analysis and record.

THE CAUSE

In one case, the patient was thrown from his horse and, in falling, one leg was severely wrenched when it remained temporarily fixed in a stirrup. In 2 other cases, men skidded on a slippery floor for a short distance, lost their balance and fell in a 'splits' position with their lower limbs spread wide apart. Another story was that of a man who also landed in a 'splits' position after tripping over a chain. The fifth case was that of a workman who fell off scaffolding, his one thigh being wrenched when it caught in a steel bar which momentarily delayed his fall.

THE MECHANISM OF INJURY

In each case, a violent abduction strain was applied to the thigh; in 4, hyperextension of the femur as an additional strain could be inferred from the description given, while in 1 of these, external rotation as a third element of mechanical force was recognizable.

CLINICAL FEATURES

Pain of immediate onset, high up the inner side of the thigh, was common to all cases. In 3 of the injuries, pain

* A paper and case demonstration presented to a clinical meeting at the Workmen's Rehabilitation Centre, Johannesburg, on 24 August 1961.

and maximal tenderness were fairly accurately situated beneath the tendon of origin of the adductor longus muscle; the tendon itself was also tender when grasped between the finger and thumb, and this sign suggested a diagnosis of sprain of the adductor longus.

Review of the anatomy of the region suggests the likelihood of pain and tenderness being situated further laterally — in fact in the femoral triangle, but this localization did not appear in this series.

In addition to the pain just distal to the groin, 4 men also had pain in the iliac fossa of the same side. There were 2 examples where, in addition, there was low back pain to one side of the midline at the level of the lumbosacral junction; radiation of pain down the antero-medial aspect of the thigh was reported in 2 instances.

In addition to local tenderness over the upper inner thigh, pain was induced in this area and also in the iliac fossa by extension, abduction and external rotation of the thigh. Four of the men held the affected thigh in a degree of flexion and walked with a limp.

In 2 cases, one within several days of the injury, and the other 4 weeks later, a swelling appeared in the upper part of the medial side of the thigh; the diffuse and tender swelling was most noticeable towards the medial side of the femoral triangle and extended deep to the adductor longus. Both of these swellings progressed to frank abscesses and were drained surgically in the belief that they were localized to the thigh. In 1 case, 9 weeks after the injury, a perineal abscess presented and discharged through 3 sinuses; this was initially diagnosed as a fistula-in-ano following upon an ischiorectal abscess. In 2 other instances, where pain and deep tenderness in the iliac fossa became the outstanding clinical evidence, a mass of poor definition became palpable deep in the iliac fossa.

Fever of irregular character was recorded in all cases, and the persistence of fever was one of the important indications for further search to find the cause. The rider injured by falling from the horse was the only one to form an abscess above the iliac crest; this followed 3 months after the abscess in the thigh. The higher abscess was drained, and a chronic discharging sinus remained. His condition was thought to be due to vertebral osteomyelitis, but repeated negative X-rays, including sinograms, finally negated this diagnosis.

This same patient presented another difficult feature; while undergoing investigation in hospital he had a severe haemorrhage per rectum. Sigmoidoscopic appearances and 'empiric' therapy suggested amoebiasis, but laboratory tests were negative.

DIAGNOSIS

There was only 1 example of the diagnosis being established radiologically. An opaque medium injected into a discharging sinus just lateral to the tendon of the adductor longus could be traced into the hollow of the ilium deep to the iliacus muscle. In the remaining cases, the diagnosis was ultimately resolved after exclusion of other suspected conditions, which included vertebral, ischial and pubic osteomyelitis, psoas abscess, rectal fistula, sprain of adductor longus with haematoma and abscess formation, femoral hernia, appendicitis with appendicular abscess, and iliac and inguinal adenitis. In 3 instances, the diagnosis, although delayed, was confidently made, but in 2 it was rather tentative, and the operative treatment was looked upon as exploratory. In 1 instance only was the diagnosis established, and definitive treatment instituted, within a week of the injury; in the remainder, the delay varied from 7 weeks to 25 months.

TREATMENT

The surgical approach is through an oblique incision, about 1 inch medial to the anterior superior iliac spine, extending for an inch or so above the level of the spine, and continuing downwards parallel to the inguinal ligament for a distance of about 3 inches. The peritoneum is not opened, but it is displaced with its contents medially, so revealing the sheathed iliacus muscle. The sheath is cut with a scalpel parallel to, and more medial than, the line of the skin incision; the muscle bundles are separated by a coarse, strong Spencer-Wells forceps, and this opening is enlarged by finger dissection, the fingers reaching bone beneath the muscle. The abscess is readily found as a distinct and recognizable entity separating muscle from bone and extending downwards towards the thigh.

Drainage of this deeply and awkwardly placed abscess is a mechanical problem. Anterior drainage is extensively impeded when the muscle bundles and the peritoneum fall back into place, and gravity cannot be brought into play to a sufficient extent. In addition to anterior drainage, posterior drainage *via* the gluteal region and through the bony plate of the ilium is established directly into the abscess cavity.

Appropriate antibiotics and general supportive treatment are also given. In all cases staphylococci were cultured; in 3 as the only organism, in 1 they were mixed with streptococci, and in 1 with *B.coli*.

RESULTS

In 1 case, that of the horserider, the follow-up has been inadequate, since the patient returned to his farm in the Eastern Transvaal and, apart from his one letter of thanks for the 'cure', he has not been heard of again. Three other patients were followed for 7 months - 1 year, and they were symptom-free during that period. The fifth patient was operated upon only a few weeks ago; he appears to be 'cured', but it is too early to be definite.

SUMMARY

A review of 5 patients who developed abscesses deep to the iliacus muscle, following injury, is given.

The mechanism of the injury, the clinical features, the diagnosis and the treatment are discussed. The difficulties in diagnosis and treatment are emphasized.

It is pointed out that the condition may not be as uncommon as is usually believed.

VAN DIE REDAKSIE : EDITORIAL

DIE BETEKENIS VAN 'N SKEPPENDE BELANGSTELLING

By die geleentheid van die Mediese Kongres wat onlangs in Kaapstad gehou is, was daar weer, soos in die verlede, 'n interessante uitstalling van die stokperdjies van dokters. As ons hierdie uitstalling volgens professionele standaarde vergelyk met ander kunsuitstallings, sal dit natuurlik minder treffend wees. Hoe kan ons dit ook anders verwag? Immers, wat ons te sien gekry het by die uitstalling van die stokperdjies van die dokters is die amateurwerk wat deur besige mense gedoen is in hul afknyptydjies.

Maar, dit is juis dít wat die belang van die uitstalling verhoog. In die tyd waarin ons leef, waarin ons daaglikse kom en gaan nou al die pas van die motor ver oorskry (ons beweeg alreeds op die projektiel-vlak en in kernbane), is dit miskien juis ons aktiwiteite in ons afknyptydjies wat die graad van ons innerlike beskawing weerspieël.

Soos Albert Schweitzer ten opsigte van ons daaglikse doen en late gesê het, dat elke mens 'n tweede taak moet hê — iets wat hy doen ten behoewe van die algemene welsyn van mense waarvoor hy nie betaal word nie, so is dit ook nodig vir ons om op die kulturele vlak 'n tweede belangstelling te hê. As dokters sal ons eerste belangstelling natuurlik moet gaan oor die kennis en insig en houdings en beginsels wat aan die grond lê van die suksesvolle beoefening van die mediese praktyk — in sy vorm as mediese wetenskap sowel as in sy vorm as lewenskuns.

Maar dan is daar nog die noodsaaklike tweede belangstelling waarna ons so pas verwys het — 'n belangstelling wat miskien geen praktiese betekenis en voordeel hoegenaamd het nie, wat dus in pragmatiese en utiliteitsterme volstrek nutteloos is, maar wat tog desnieteenstaande 'n weerspieëling is van daardie ander en dieper dors wat die wesenlike mens nog altyd gekenmerk het.

Watter spesifieke vorm hierdie tweede belangstelling aanneem, maak nie eintlik saak nie. Dit kan byvoorbeeld een of meer van die vorms aanneem van die stokperdjies wat ons op die uitstalling by die Kongres te sien gekry het — die versameling en herbelewing van amulette, skulpe, vetplante; of plastiese werk, beeldhouwerk, skilderwerk, kunsfotografie, ens.

Andere sal ander belangstellings hê. Charles Darwin het byvoorbeeld gesê: ‚As ek my lewe weer kan oorlewe, sou ek 'n reël maak om minstens een keer in die week 'n bietjie poësie te lees en na musiek te luister; want dan miskien sou daardie dele van my brein wat nou verdor is, lewendig gebly het deur gedurige gebruik. Die verlies van die smaak vir hierdie dinge is die verlies van geluk. Die verlies daarvan benadeel heel moontlik 'n mens se intellek. Baie waarskynliker benadeel dit 'n mens se sedelike karakter deurdat die verlies van hierdie dinge die emosionele deel van ons menslike natuur verarm'.

En E. M. Forster het in sy treffende essay oor ‚Anonimiteit' in *Two Cheers for Democracy*, gesê: ‚Lyric poetry is absolutely no use, and poetry generally is almost no use ... What's the use of "a slumber did my spirit seal" or ... "so we'll go no more a-roving"? ... Imagination is our only guide into the world created by words ... What there is down there — ah, that is another enquiry, and may the clergymen and scientists pursue it more successfully in future than they have in the past'.

Wat die vorm van ons tweede belangstelling is, maak dus nie so veel saak nie. Wat van belang is, is dat dit daar is en dat dit kultureel van soort en skeppend van aard is. ‚Die mens wat iets skep' — so het so 'n voortreflike beoefenaar van die lewenskuns, soos M.E.R., in ‚Ou skuld' in *Die Gewers*, gesê: ‚die mens wat iets skep — hy vergeet baie van die swaar en die skades wat hom oorkom'.

In die uitvoering van die opdrag: ‚medisynmeester, genees jouself', speel die aankweek van 'n skeppende belangstelling, bowe en behalwe die daaglikse verpligtings van professionele arbeid, dus 'n belangrike en essensiële rol. Maar ook vir ons pasiënte is hierdie kennis en insig van die allergrootste belang, en elke dokter wat self die verruimende en bevrydende uitwerking van 'n skeppende belangstelling leer ken het, sal dit op oortuigende wyse kan oordra aan sy pasiënte, nie net as 'n *voorskrif* vir 'n gesonde en gebalanseerde lewe nie, maar ook en veral as 'n voorwaarde vir die geestelike groei en ryping van die eintlike, wesenlike mens. Dan sal hy sowel as die pasiënte wat aan sy sorg toevertrou is, ook in staat wees om die werklike betekenis van hierdie woorde van die digter te snap:

> ‚Want zie, zoo lange tijd is aan elk mensch gegeven
> Dat zijn woord rijpe tot lied
> Voor Gods aangezicht'.

THE LANGUAGE OF THE PATIENT

In the course of his Presidential Address, which was delivered at Cape Town during the recent Medical Congress, Mr. Currie, President of the Medical Association of South Africa, said: 'One desideratum in an educated man is the gift of being explicit and articulate in language. In a bilingual country such as ours, the really perfectly bilingual have so great an advantage in mental agility and elasticity that they are to be greatly envied, provided that the trap of mediocrity in both languages can be avoided. This projection into another language should usually make it easy to have some acquaintance with a third, and I feel that doctors should learn one of the major European languages for its interest, its literature, and the access it gives to appropriate medical publications. To this polyglot equipment might well be added a basic knowledge of the Bantu tongue most prevalent in the area'.

This considered pronouncement of the President expresses the same basic sentiments that are voiced in the impassioned plea of a colleague from Durban (published on p. 1000 of this issue of the *Journal*), that every doctor in the country, practising among non-European patients, should be able to talk and understand the language of his patient. Dr. Campbell summarizes his own thoughts in this connection by paraphrasing a sentence used by Alan Paton at the Natal Medical Graduates Dinner in October 1961, in the following words: 'We should be ashamed that, as the profession least conscious of race in this country, so few of us working among non-European patients have made the slightest attempt to learn the language of the patient, or a language mutually known to ourselves and the patients'.

We feel that we must support these sentiments without any reservation whatsoever. We should even like to go a step further and state categorically that it is morally wrong for any doctor to practise 'for gain' among people unless he is able to communicate direct with them in a language which they understand well enough to express accurately, not only the overt symptoms of their physical illnesses, but also the finer nuances of their thoughts and emotions, and the true nature of their hidden hopes and fears.

We all know the numerous 'humorous' stories in circulation about the inability of interpreters, in the courts of law, to convey to the Judge the true meaning and implications of the words of the accused, and *vice versa*. These, often facetious, stories reflect an unsatisfactory reality which, in the practice of medicine (if not in the administration of Justice — but that is fortunately not primarily our responsibility), may well lead to a lowering of our standards of diagnosis and treatment — not to mention the level of our interpersonal and intercultural relationships.

In the practice of psychiatry, for instance, it is imperative and obligatory for the therapist to be able to establish a satisfactory *rapport* between himself and his patient, and also to have the ability to enter into a true empathic experience. A knowledge of the patient's language or a language mutually known to the patient and his doctor, is a prerequisite for achieving these two basic requirements for a satisfactory doctor-patient relationship in psychiatric practice. Without this we have no hope of ever overcoming the already formidable barriers to transcultural communication.

All this is bad enough when Europeans have to be treated by fellow Europeans who do not understand 'the language of their hearts'. It is infinitely worse when the needs of every person in South Africa is considered, especially in view of the fact that there is not a single, trained non-White psychiatrist in the country. For years to come it will therefore inevitably have to be the White man's duty and privilege to take a lead in building the necessary empathic bridges.

These considerations are important, not only in the practice of psychiatric medicine, but also in the practice of medicine in general. In spite of the fact that it is relatively easier (in certain instances) to treat physical conditions on the basis of diagnoses made on objective signs, our margin of error remains dangerously high (and the level of our standards dangerously low) if we have to rely on secondhand information gained from interpreters who have had insufficient training and experience in both medicine and the humanities to enable them to act as intermediaries between vague and apprehensive patients, on the one hand, and harassed and impatient doctors on the other.

It is of course obvious that true bilingualism or multilingualism will always remain outside the bounds of realization for most people — a situation which, in this country, is complicated beyond comprehension by the welter of Bantu languages. Many people, belonging to all racial groups, are in any case sadly lacking in the ability to communicate their thoughts on an articulate and explicit level. There are, in fact, people who are practically inarticulate in any language — an observation which long ago was symbolized by Fitzgerald in his superb translation of Omar Khayyam:

> 'And strange to tell, among that Earthen lot
> Some were articulate, while others not . . .'

This very fact imposes an even greater obligation on us to approach this essentially human problem of the language of the patient on a mature and responsible level and in a truly understanding spirit.

SHOULDER-ARM PAIN*

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The question of shoulder-arm pain is, perhaps, the most confusing of subjects, and one of those still largely unsolved in medicine and surgery. In an attempt to illustrate the problem, this paper considers the possibility of positions and movements causing compression of the neurovascular tree because of anatomical factors. Included are skeleton studies, a study of personal symptoms, and the application of conclusions to an assessment of clinical cases. A review of the historical development of the views on this problem is given.

* Extracted from a paper delivered at the Second Congress of the Association of Surgeons of South Africa (M.A.S.A.), Durban, 17 - 20 September 1960.

SKELETON STUDIES

The neurovascular tree was reconstructed and the scalenus anterior and pectoralis minor placed in relation to the tree (Fig. 1). It is, of course, impossible to claim that this truly represents what occurs in life. Movements caused dramatic alterations in the relations of the reconstructed

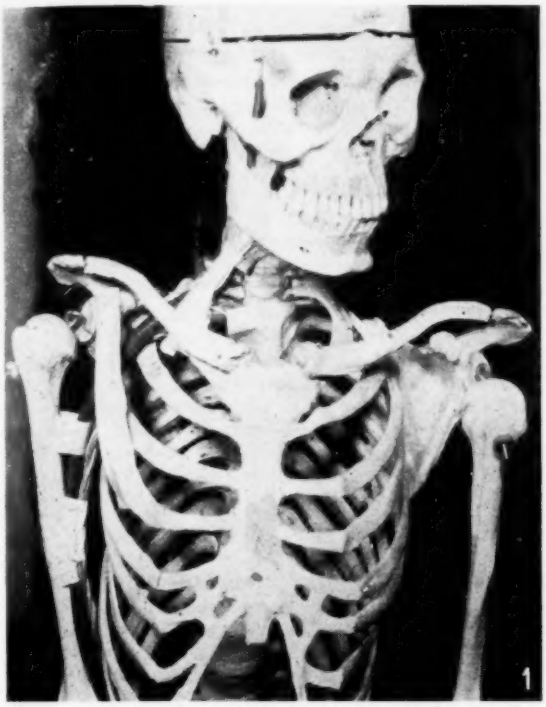

Fig. 1. Skeleton with reconstructed neurovascular tree in right arm.

tree to the anatomical structures, and it is at least possible that this may happen to some extent in the living person.

Scalenus Anterior and Medius

Information from these studies showed that, on elevating the shoulder towards the head, the neurovascular bundle is pulled against the edge of the scalenus anterior (Fig. 2). How much movement is permitted in life is difficult to say, but when the movement is allowed in the skeleton this is most noticeable. There is certainly a jamming upwards between the scalenus anterior and medius.

Pectoralis Minor

Further, on forward elevation of the arm, while keeping the shoulder absolutely level, the neurovascular bundle did definitely kink at the level of the pectoralis minor muscle. On abduction of the arm without elevation of the shoulder, there is a tautening of the neurovascular bundle with some kinking at the pectoralis minor.

A combination of elevation of the shoulder and abduction of the arm affords a double mechanism of tautness and kinking, with pressure on the scalenus anterior or in the wedge between the scalenus anterior and scalenus medius (Fig. 3). The pectoralis minor affords a second point of pressure from angulation and tautness beneath it.

Traction on the Neurovascular Tree

Studies were also made, on the skeleton, of tensions and tractions on the neurovascular tree. It is well known that traction injuries occur, e.g. as when the shoulders and the

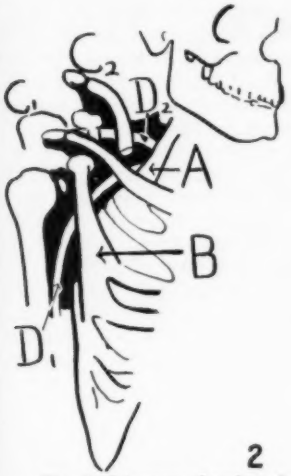

Fig. 2. Diagram showing shoulder elevation (C_1 - C_2) with elevation of neurovascular tree (D_1 - D_2). (A = scalenus anterior, B = pectoralis minor.)

Fig. 3. Diagram showing elevation and abduction of shoulder (C_2 - C_1) with elevation and kinking of taut neurovascular tree (D_1 - D_2). (A = scalenus anterior, B = pectoralis minor.)

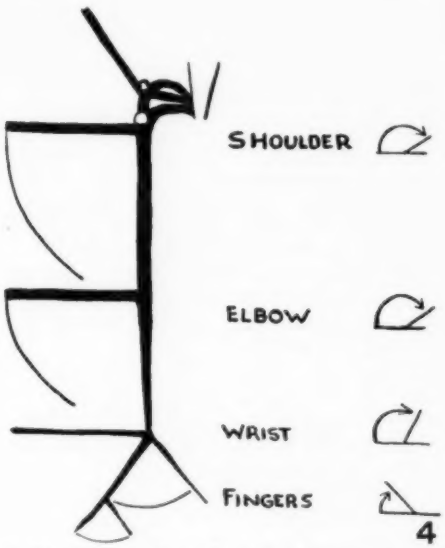

Fig. 4. Diagram showing tautening of neurovascular tree. Movements from flexion to extension at elbow and wrist. Abduction of shoulder.

head are separated in a fall on the shoulder, resulting in a complete or partial tear of the brachial plexus, often at Erb's point.

A young doctor jumped over a tennis net and fell on his extended arm with the hand and fingers extended as well. He immediately suffered pain in the shoulder, and this was followed by pain in the areas of the anterior divisions of the nerves C 5 and 6. This lasted for several weeks, and was obviously from traction injury of the neurovascular tree.

In the case of the skeleton, fishing line was used. The plexus was fashioned to run under a reconstructed scalenus anterior muscle into the arm, under a reconstructed pectoralis minor muscle, and then under a reconstructed carpal tunnel to the tips of the fingers. Loose loops were used to maintain the reconstructed vascular tree in the fingers (to copy the action of the flexor tunnels) and were also used in the forearm and in the region of the humerus. A tensionometer was used, which was the facepiece and lever of an oscillometer, in which a cotton attachment to the needle allowed the dial to be adjusted to a standard position. Extension of the elbow from a semi-flexed position (a right-angle flexed position), to full extension, and abduction from the position of the arm by the side to 90° abduction, was studied. The results indicated that quite considerable traction and tension occur with these movements (Fig. 4).

Use of the Tensionometer

Extension of wrist and fingers. On fixing the indicator of the dial to 0 and adjusting the manometer to a standard position which was present at full flexion of the wrist, it was found that extending the wrist and the fingers fully caused the tensionometer to record in the region of 130 mm.Hg.

Extension of the elbow. The investigation was applied also to the elbow and shoulder and, from right-angle flexion to full extension of the elbow the needle was displaced to the full scale of the manometer and would probably have gone further if possible.

Abduction of the shoulder. This same experience was obtained on abducting the shoulder. It was thus possible to appreciate roughly the amount of traction that is exerted on a fixed point in the distal forearm by extension of the elbow and abduction of the shoulder.

Comment. Although these experiences are crude and there is difficulty in comparing these findings with what happens during life, there is no doubt from this simple experiment that a considerable traction effect on the neurovascular tree does occur during these movements. Kinking, and traction and pressure are possible from the taut kinking at the scalenus and pectoralis minor muscles in extension and abduction.

The wrist. Sunderland,[1] in 1945, carried out an interesting experiment. He injected the brachial artery in a fresh cadaver and was able to show the blood supply in the median nerve. On flexion of the wrist, an area corresponding to the carpal tunnel showed no blood supply in the nerve. Obviously, pressure sufficient to occlude nerve vessels occurs on wrist movements.

Discussion

It is apparent that tautness, kinking and pressure are common factors in the aetiology of shoulder-arm pain, apart from pathological causes of neurovascular abnormality. Normal movements will produce fleeting attacks of pain, whereas occupational or forced maintenance of abnormal positions and the prolonged effects of position when asleep may be the determining factors.

Abnormal anatomy, such as fallen shoulder-tip in costoclavicular compression, cervical ribs, a highly placed subclavian artery, etc., will play a part in postural decompensation.

It is known that the excision of the clavicle has relieved clinical costoclavicular symptoms when scalenectomy has failed. A case of this nature was presented recently by Prof. D. J. du Plessis in Johannesburg. In this patient relief had been afforded by leaning forwards. There was also night numbness and pain. On X-ray examination, osteophyte encroachment had been demonstrated, as well as notching on the under medial portion of the clavicle. Removal of the major pressure by excision of the clavicle gave dramatic relief even when psychiatric advice had been against the operation.

PERSONAL SYMPTOMS

Schultze[2] originally described the syndrome of numbness and paraesthesia of the hands, associated with certain positional alterations, as acroparaesthesia. Being a mild sufferer from these symptoms myself, I made observations on them, and attempted to correlate the signs and symptoms with the supposed pathology, and investigated symptoms developing in different positions. A proved encroachment of cervical intervertebral foramina was present, and its relationship to the development of symptoms will be described at a later stage.

Symptoms Attributable to the Scalenus Anterior

The most typical symptoms which I attributed to scalene pressure were those occurring on sleeping on the affected side. Numbness in the arm, hand and fingers disturbed sleep. The symptoms were relieved by turning over to the other side. It was thought that the brachial plexus was elevated against the edge of the anterior scalene with pressure or traction effects. The following is a typical case history:

A woman, aged 58, had symptoms for 33 years following a fall from her bicycle, in which she injured her shoulder. This was diagnosed as 'brachial neuritis', presenting with numb 'dead' hands; waking in the night with 'dead', numb and painful hands; and inability to write or knit. Symptoms could be reproduced by lying on her side with shoulder-head approximation.

X-rays of the neck showed no cervical spondylosis or cervical ribs. She had seen numerous doctors for this condition. She had been treated with vitamin-B_{12} injections, X-ray therapy, injection of fibrositic nodules, neck traction, and ACTH, and had had treatment for tennis elbow.

Operative division of the scalenus anterior revealed a high subclavicular artery, and, after freeing this and the plexus, symptoms were completely relieved (follow-up 7 years).

Other similar results have been obtained from operative division of the scalenus anterior muscle and freeing the plexus in acroparaesthesia with night numbness. Degenerative changes have been noted in the brachial artery and the scalenus anterior muscle; these were mentioned by Oschner and de Bakey.[3] Other patients with similar symptoms in the presence of cervical ribs have obtained relief following resection of the ribs and scalenectomy. Careful assessment of symptoms in shoulder lesions has revealed further examples of plexus-scalene pressure.

Two patients suffering from fairly long-standing painful supraspinatus tendinitis later developed typical night numbness and tingling in the hand and arm, explainable only on a plexus-pressure basis. This was assumed to be the result of the characteristic protective position that is taken up in these cases. The shoulder is elevated towards the head, with or without the use of a sling. Relief of

the cause of pain and restoration of shoulder function followed treatment of the supraspinatus tendinitis.

Symptoms Attributable to Pectoralis-minor Pressure

On driving a motor-car with my arms extended straight in front and gripping the top of the driving wheel, symptoms of 'dead', numb fingers occurred.

Wright,[4] in describing the hyperabduction syndrome, an acroparaesthesia of occupational nature in riveters and ballet dancers, etc., regarded the pectoralis minor as an important pressure point associated with costoclavicular pressure.

It was thought that the symptoms following elevation of the arm holding the upper part of the wheel were almost certainly from pectoralis-minor pressure. However, the study was carried further. Symptoms in the left hand and arm invariably began when the arm was extended in front on the top of the steering wheel, with the clenched fingers (flexor digitorum sublimus contracted) and the thumb pressing firmly against the wheel (thenar-muscle contraction and carpal-tunnel tension). The numbness seemed, under these circumstances, to be in the distribution of the median nerve. Immediately the hand was rested on the wheel without gripping, the symptoms disappeared. I could only assume that what I had thought to be pectoralis-minor pressure was in fact carpal-tunnel pressure. The unreliability of explanations was thus obvious and portended great difficulties in the future study.

Another disconcerting problem arose when the same position on the driving wheel was taken up, i.e. forward elevation of arms and semi-extended elbows, fingers flexed and thumb pressing on the wheel, but with the forearm supinated instead of pronated. Then no symptoms could be reproduced. Immediately the pronated position was restored, symptoms recommenced, and the moment the forearms were supinated the symptoms disappeared again. This could only mean that the pronator teres had to be brought into action in these circumstances for symptoms to appear. As the median nerve passes between the 2 heads of the pronator teres, these muscles partake in the production of symptoms.

Symptoms Caused by Local Pressure

On rare occasions the wearing of a tight rubber glove caused numbness in the thumb. Also, leaning over a chair caused pressure on the medial side of the upper arm with resultant numbness in the hand. A strap over the shoulder occasionally did the same thing.

Symptoms Attributable to Head and Neck Movements

These were rather vague and localized to the posterior part of the neck and shoulders, prominent when high pillows were used, or during a visit to the planetarium.

Symptoms Caused by Costoclavicular Pressure

This pressure was thought to cause the occasional numbness that occurred with the arms by the side when carrying weights, etc. This did not constitute a major problem.

Symptoms Attributable to Pressure at Wrist Level

Many tests were carried out to see whether wrist movements in any particular position caused the symptoms in the fingers. At no time was it possible to say definitely that wrist flexion or extension alone caused symptoms in the hand. Tests were carried out in patients as well, who were asked to sleep holding a newspaper to keep the hands from flexing or extending. Only one patient attributed symptoms to extension of the wrist from holding a telephone for long periods or pushing a pram up a hill, as well as to waking up at night with the wrist fully extended under the chin. This patient was a young housewife who also complained of night paraesthesia. According to Walshe this was a typical costoclavicular syndrome, and according to Nissen it was a typical carpal-tunnel syndrome. Activity of the hands was associated with symptoms in a few patients.

HISTORICAL SURVEY

Before 1947

This was the 'golden era' of scalenectomy. Scalenectomy was all that was necessary, even in cervical rib abnormalities.[5] The scalenus-anticus syndrome was recognized[2,6] and, in fact, histological signs of degeneration and inflammation were actually described.[3]

The Debunking Era

About 1947 there was a period which could be called the 'debunking era' of scalenectomy. Telford and Mottershead,[7] in 1947, showed that the Adson arterial-obstruction tests of head and neck movements, and deep breathing obstructing the pulse, occurred in normal subjects. They stressed that there was no agreement as to the cause of the compression of the brachial plexus. The scalenus medius was implicated as well, and direct traction on the nerve roots was a factor in certain circumstances. Cervical ribs and their remnants, according to these writers, still accounted for the largest proportion of cases.

At this time, too, Brain, Wright and Wilkinson[8] described the division of the carpal tunnel in 6 well-selected cases of median-nerve compression. The focus of interest was passing from the thoracic outlet to the wrist. There were, however, other workers interested in the thoracic outlet.

The Costoclavicular Syndrome

Falconer and Weddell,[9] in 1943, described the costoclavicular syndrome of compression between the clavicle and the ribs, referring mainly to arterial compression. Walshe *et al.*[10] strongly supported the view that acroparaesthesia in middle-aged women was caused by the pressure of the plexus against the first rib following muscle decompensation and loss of tone (Fig. 5). During the War middle-aged women found that they had more unaccustomed work to do. This syndrome was also described in pregnancy and in the nursing mother. It was also caused by unaccustomed muscular strain and increased activities in patients lacking in general muscular tone. The shoulder

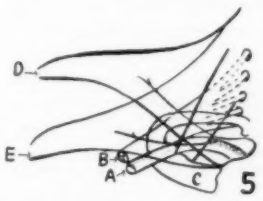

Fig 5. Diagram showing pressure of the brachial plexus and subclavian artery against the first rib with a fallen shoulder tip, the result of muscle decompensation and loss of tone. (A = subclavian artery, B = brachial plexus, C = first rib, D = normal shoulder tip, E = fallen shoulder tip.)

tip was consistently found to be lower than the sterno-clavicular joint.

In my series, costoclavicular compression was regarded as important in several cases. In 2 patients, fractured clavicles with bosses of callus causing pressure on the plexus brought on these symptoms on walking, carrying bags, and depressing the shoulders and retracting them backwards. These symptoms did not cause the typical night paraesthesia.

Two males got the symptoms lying in bed after abdominal operations. In each case the neck was flexed by too many pillows, the arms hung down at the sides, and the patients could not turn on their sides. The symptoms were relieved when the patients became ambulant and were able to sleep on their sides.

The Hyperabduction Syndrome

In 1945, Wright, a colonel in the American Army, investigated what he called the 'hyperabduction syndrome',[4] a syndrome of occupational nature in which riveters, ballet dancers, and other workers with arms elevated above the head had typical symptoms of acroparaesthesia. He studied normal subjects and came to the conclusion that the compression of the neurovascular tree arose from costoclavicular compression together with pectoralis-minor compression. Spontaneous axillary thrombosis has been linked with this positional state and with pectoralis-minor pressure, but radiologically it has also been shown to occur following pressure from the scalenus anterior muscle.

Cervical Spondylosis

During the period 1946 - 1956 the syndrome of cervical spondylosis became recognized. At first cervical discs, like lumbar discs, were blamed. Later, encroachment of the intervertebral foramina and osteophytes were considered the major cause. Patients with acroparaesthesia without wasting or neurological signs often respond to neck traction.

This has not been the experience of a neurological colleague, and I can only assume that his patients have obvious neurological signs and wasting, whereas my patients have presented only with night paraesthesia.

It was found that true nuclear herniations in the cervical region are rare[5] and the diagnosis of motor-neurone disease, syringomyelia, spinal disseminated sclerosis, and progressive lateral sclerosis are considered by some to be entities apart from this condition of cervical spondylosis, whereas others consider them to be caused by different selective sites of compression. Osteophyte formation is recognized as a cause of local pressure and the nerve roots may be affected by direct pressure or ischaemia. Canal capacity and root-sleeve fibrosis are important factors as well.

That acroparaesthesia occurs in a large number of patients with cervical spondylosis with encroachment is undoubted. I myself have the typical night numbness and tingling. Why should symptoms from this condition be apparently identical with those in patients suffering from cervical-rib or scalene syndrome, and even carpal-tunnel syndrome?

It is thought that segmental spasm of the scalene could be the cause of 'secondary' scalene symptoms, or it could be that the pathology in the neck makes the nerves more susceptible to pressure effects which would not be noticeable in a normal subject. Can it be that the state of the major nerve roots is one factor, and that pressure points and traction effects pertaining to several anatomical sites can be other separate factors?

Carpal-tunnel Syndrome

The carpal-tunnel syndrome was well known even in 1909, when Hunt[11] described division of the carpal tunnel and found a neuroma under it. Marie and Foix[12] described similar cases in 1912 and proved one by necropsy dissection.

Brouwer,[13] Harris,[14] Lhermitte and de Massany,[15] Dorndorf,[16] Moersch,[17] Wartenburg,[18] Woltman,[19] and Zachary[20] all contributed to knowledge on this subject, and Brain, Wright and Wilkinson,[6] with their 6 cases of operative division of the carpal tunnel, popularized this diagnosis.

We are all occasionally confronted with obvious cases of median-nerve involvement in the hand, with thickening and neuroma formation under the carpal ligament, associated with sensory abnormality and wasting of muscles supplied by the median nerve.

The extension of the operation dividing the carpal tunnel to the treatment of a large number of patients with acroparaesthesia is, however, a new concept. Nissen[21] wrote: 'Division of the transverse ligament of the carpal tunnel for carpal-tunnel compression of the medial nerve, thanks to the warm advocacy of the Middlesex School, has become by far the most frequent of all operations for the relief of pain in the upper arm'.

Nissen[22] also wrote: "Since 1947, at the Nuffield Orthopaedic Centre at Oxford, this operation has become a common one and the volume of patients requiring decompression justifies a procedure being performed in the outpatients' department, under local anaesthetic".

I have known of physicians who state that in their experience at least 80% of patients with these symptoms are relieved by division of the carpal tunnel, and they seek out trained surgeons to perform the operation. The indication is no longer the obvious median-nerve lesion with wasting and sensory changes in the distribution of median nerve, associated with a neuroma under the carpal ligament, but the operation is urged in patients with the syndrome of acroparaesthesia, even when the distribution of pain and numbness extends far above the arm, even into the shoulder.

As one who has seen the different eras and developments in this syndrome, I am not so easily converted to this line of thought. I can quite willingly accept that division of the carpal tunnel does relieve a large number of patients. It is difficult, however, to explain certain features. Why is night pain typical of this disorder when it is also typical of other lesions? Why also, when a sphygmomanometer cuff is blown up to 200 mm.Hg. causing tingling within 1 minute in the hands and the fingers in the median-nerve distribution, is this considered diagnostic of the carpal-tunnel condition, when tingling of this nature occurs in many lesions?

It is difficult to accept the statement of Nissen,[21] who said: 'Clear thinking on the subject is essential since there are two definite types — (*i*) the spontaneous primary compression which occurs in females, usually middle-aged

females; (*ii*) secondary compression from early rheumatoid tenosynovitis, long-standing carpal-bone lesions, acromegaly, and fluid retention such as occurs in pregnancy and myxoedema'.

It would seem that even clearer thinking is required to explain the empirical relief obtained by carpal-tunnel division and its use on a wide scale for lesions not necessarily in the distribution of the median nerve.

CASE HISTORIES AND DISCUSSIONS

The following specially selected cases are described to demonstrate problems arising in the shoulder-arm syndrome:

Case 1 — Mrs. H., Aged 58 Years

This patient had a scalenectomy for acroparaesthesia and night numbness caused by sleeping on her sides. Relief was obtained. This demonstrates that the operation helps in this condition, and also shows how a high subclavian artery, bunching the plexus under the scalenus anterior muscle, can be a factor in causing this syndrome.

Case 2 — Mrs. W., Aged 44 Years

This patient had Raynaud-like symptoms and night-numb-pain for 1 year, with blanching of fingers and sleepless nights from pain, as well as numbness on sleeping on her sides. X-rays were normal with no evidence of cervical ribs or cervical spondylosis.

Complete relief of the night-numb-pain and the vascular symptoms followed scalenectomy, freeing of the neurovascular bundle, and bilateral cervical sympathectomy. The subclavian artery was higher than normal at its origin.

Discussion

The co-existence of Raynaud's syndrome and brachial-plexus compression symptoms focused attention on the scalene muscle region. Relief of all symptoms followed scalenectomy.

Case 3 — Mrs. M., Aged 32 Years

This patient was relieved of 5 years of night-numb-pain by scalenectomy. There had been no evidence on X-ray of cervical ribs or encroachment of the intervertebral foramina. Bilateral high subclavian arteries were found. A physician treating the patient had asked for a carpal-tunnel release operation to be performed, but this was done only on the right side, whereas a bilateral decompression of the neurovascular tree and scalenectomy were performed.

After the operation symptoms were relieved on both sides; some recurrence occurred after 6 months on the right side. Knitting had been impossible until the carpal-tunnel release operation had been performed.

Re-exploration of the right supraclavicular region and freeing of the artery and the nerve plexus resulted in relief of the recurrent symptoms, and 1 year later the patient was still free of symptoms. At the second operation the artery on each side was adherent to scar tissue, and the plexus, especially on the right side, was being displaced posteriorly by a fibrous band.

Discussion

This patient was to some extent a test case. The physician diagnosed the carpal-tunnel syndrome. No swelling of the median nerve was visible on exposure beneath the right carpal tunnel. Further, symptoms were bilateral and relieved on both sides by scalenectomy and decompression, although carpal-tunnel release was only performed on the one side. Recurrent symptoms were relieved immediately by release of scar tissue in relation to the subclavian artery and the nerve plexus.

Case 4 — Miss G., Aged 34 Years

Following an attack of encephalitis, the patient was left with residual shoulder-arm pain and a typical night-numb-pain syndrome. On the right side the symptoms were so severe that sleep was lost. Relief was sought following continuance of the pain after deep-X-ray therapy to the spine, and 8 months' conservative treatment without improvement.

There was a history of numbness when the arms were extended on the steering wheel while driving a car, and a typical middle-of-the-night syndrome on lying on her side.

After the anterior scalene and the pectoralis minor were divided on the worst side, symptoms were dramatically improved. Only some residual numbness occurred on using the fingers and hands, but no further night-numb-pain.

Discussion

The occurrence of the typical syndrome of acroparaesthesia in a radiculitis following encephalitis was noted and the

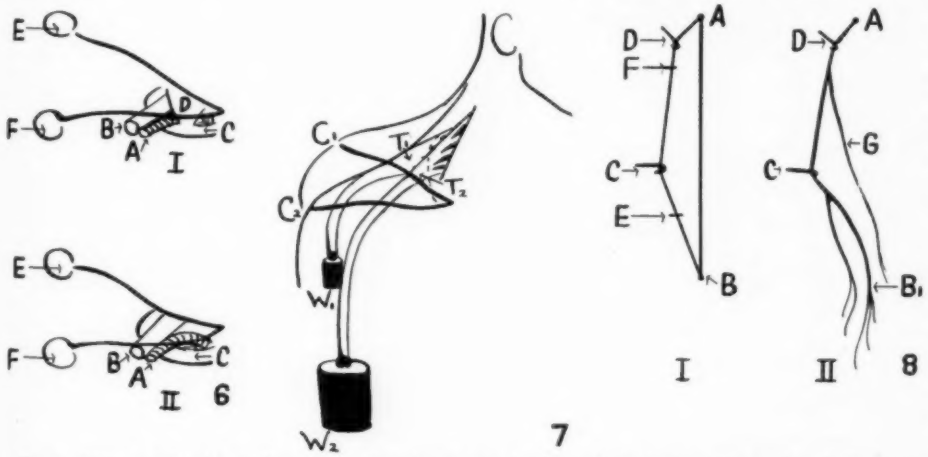

Fig. 6. Diagram showing buffer action of scalenus anterior in costoclavicular compression. I. Scalenus anterior present. II. Scalenus anterior removed. (A = subclavian artery, B = brachial plexus, C = first rib, D = scalenus anterior, E = normal shoulder tip, F = fallen shoulder tip.)

Fig. 7. Diagram showing traction on brachial plexus as a result of a fallen shoulder tip. (C_1 and C_2 = positions of shoulder tip, T_1 and T_2 = brachial plexus, W_1 and W_2 = representation of degrees of traction.)

Fig. 8. Diagram showing the effect of carpal-tunnel release on the neurovascular tree. I. Carpal tunnel intact. II. Following carpal-tunnel release operation. (A = fixed point at the root of the neck, B = fixed point in certain positions at carpal tunnel, B_1 = effect of carpal-tunnel release, C = effect of flexion of elbow, D = effect of abduction at shoulder, E = pronator teres point, F = pectoralis minor point, G = radial nerve, which skips the carpal tunnel.)

secondary part played by the scalenus anterior and pectoralis minor was established by these observations.

Case 5 — Mrs. D., Aged 25 Years

This patient was a nursing mother with a 3-month-old baby. She had typical acroparaesthesia and night-numb-pain in her hands, arms and shoulders, which woke her at night. X-ray of the neck had shown no cervical ribs or spondylosis. Neck traction had not relieved the symptoms. The cuff test showed no typical findings, i.e. the numbness would come on late and in the ulnar-nerve distribution as well as medially. Pushing a pram and holding heavy objects would bring on symptoms.

A bilateral scalenectomy relieved the symptoms immediately, but within 2 months she had a recurrence of night-numb-pain in the hands. Scalenectomy had removed all arm and shoulder pain, but not the hand symptoms. Bilateral carpal-tunnel release then relieved all symptoms including the night-numb-pain. No evidence of nerve thickening or neuroma was seen under the carpal tunnel.

Discussion

The features of this case fit into the syndrome of costoclavicular compression of Walshe, but they are also typical of the carpal-tunnel syndrome, according to Nissen's standards.

The recurrence of night-numb-pain after scalenectomy had not yet been observed to any extent except for case 3, where re-exploration and freeing of the subclavian artery and plexus from adhesions was followed by relief. Here, however, recurrence occurred, but carpal-tunnel release cured the symptoms. The reason for this could be one of the following:

1. The irritable neurovascular tree was still subject to costoclavicular compression following scalenectomy, in view of the absence of a buffering scalenus anterior (Fig. 6). More traction is exerted on the plexus with a fallen shoulder tip (Fig. 7); in this case the protecting action of the scalenus anterior would be even more important.

2. Carpal-tunnel release so relaxed the neurovascular tree that the compression at the scalene level no longer mattered (Fig. 8).

3. The original cause of the nerve irritability (? oedema, ? calcium deficiency) and increased muscle tone had disappeared at this stage of recovery.

Case 6 — Mrs. S., Aged 44 Years

This patient is a diabetic and suffers from a diabetic neuritis and tenderness in the legs as well as severe acroparaesthesia in the arms. For 5 years she had night-numb-pain symptoms with lack of sleep, and symptoms on driving a car.

A small cervical rib was present on the right side. Bilateral scalenectomy decompression and removal of the rib remnant relieved her symptoms for 6 months, but at the end of 2 years, because of recurrent night-numb-pain symptoms, a bilateral carpal-tunnel release operation was performed as recommended by her attending physicians. There had been only partial relief from neck traction.

Following operation she complained of burning in the median-nerve distribution, and also on the finger tips of the ring and little fingers. Symptoms in the right arm and hand were aggravated, whereas the left side was almost symptomless.

Discussion

The probability of diabetic neuritis as one of the factors in the abnormality of the neurovascular tree had to be considered in this case. Recurrence of night-numb-pain symptoms after relief for 6 months suggested either: (1) adhesions; (2) recurrence of cervical rib growth; or (3) a costoclavicular syndrome secondary to scalenectomy, whereby the rib and clavicle would impinge directly on the neurovascular tree without the intervention of the scalenus anterior 'buffer'.

A burning median-nerve neuritis with mild depletion of sensation occurred in case 7 as well. These 2 patients both complained tremendously regarding this. Where immediate and dramatic relief is considered a *sine qua non* of carpal-tunnel release, this complication was most disconcerting.

After about 2 months the symptoms gradually improved and the final result has been relatively satisfactory.

Case 7 — Mrs. R., Aged 58 Years

This patient had indisputable cervical encroachment with symptoms of acroparaesthesia, night-numb-pain, and disturbed sleep. There had been no relief from neck traction and a physician and surgeon had both previously agreed that a carpal-tunnel release operation was indicated; they referred the patient to me.

The cuff test on the right side was positive, but on the left the tingling was somewhat delayed and was also disturbed in the ulnar nerve region. A ganglion was visible at the wrist on the right side, extending under the carpal ligament, and there was some osteoarthritis of the wrist joint.

A carpal-tunnel release operation was followed by the appearance of a mild, hot swelling which was attributed to the reaction, especially in the degenerated tissues in relation to the ganglion wall. It remained for a long time. A mild hypoaesthesia and a persistent intolerable burning were present in the distribution of the median nerve. Symptoms were worse on the right side and mild early Dupuytren's contraction became visible in the palms.

Treatment with antibiotics, physiotherapy, deep X-ray therapy and hydrocortisone all failed to help the patient. It was even considered that a possible psychoneurosis was the cause. The swelling slowly subsided, but the patient still complains of pain and burning, especially when she uses her hands and carries anything.

Discussion

This patient had osteoarthritis of the spine with encroachment of C 5, 6, and 7, but, according to clinical tests, she was diagnosed as having a carpal-tunnel syndrome with an associated ganglion. Wearing gloves caused a numbness of the dorsum of the thumb. After carpal-tunnel release there was a burning neuritis of the median nerve, and no relief of symptoms.

This case illustrates the fact that carpal-tunnel release is not a universal panacea for acroparaesthesia on the grounds suggested. Diabetics and patients with cervical osteoarthritis present a special problem.

Case 8 — Mrs. W., Aged 41 Years

On 14 July 1960 this patient presented with a history of 3 weeks' numbness and pain, with night-numb-pain in the hands and arms causing loss of sleep. The radiation was in the median-nerve distribution, and the tourniquet test was positive. X-ray showed no encroachment on, or narrowing of, the intervertebral spaces. However, a small, left-sided cervical rib was present, as well as a long transverse process of C 7 on the right side. Operation was advised.

On 9 August she fainted and cricked her neck. Since then there has been no further trouble.

Discussion

This case demonstrates how acroparaesthesia with typical night-numb-pain can be relieved by a crick of the neck. It would be almost impossible to call this lesion a carpal-tunnel syndrome, in spite of the diagnostic similarity of signs and symptoms, based on the 'modern' concept.

In a class of 18 students, 6 admitted to night-numb-pain symptoms on lying on one or other side. Such a common set of symptoms associated with shoulder-head

approximation is unlikely to be caused by carpal-tunnel compression, in spite of the claims that this syndrome is typical of that lesion.

CONCLUSION

From this study it would appear that:

1. Symptoms of acroparaesthesia can result from several different factors.

2. Tautness, kinking and pressure of a prolonged nature, either from occupation or from position in sleep can cause symptoms by involvement of several anatomical points.

3. Altered anatomy, e.g. cervical ribs and bands; a high subclavian artery; relative postural decompensation following changes caused by operations; etc., will aggravate these factors.

4. Debunking of scalenectomy is unnecessary. There is some justification for scalenectomy or for palmar release in selected cases, and a unified concept embraces the historical complexity of the problem.

5. Pathological irritability of the neurovascular tree may be from medical causes, and radiculitis, diabetes, arteriosclerosis and oedema, for example, must not be overlooked.

6. Night-numbness and pain from lying on the affected side, and waking in the early hours of the morning from these symptoms, seems most frequently to be caused by scalenus-anterior pressure on the neurovascular tree, but relief is sometimes afforded by attack on apparently unrelated structures, e.g. excision of the clavicle and carpal-tunnel release.

7. The scalenus-anterior buffer is described, and the possibility of aggravation of costoclavicular symptoms after division of the scalenus should be borne in mind.

8. The shoulder-elevation factor is suggested as the explanation for secondary acroparaesthesia, night numbness and pain arising in patients who have had a painful shoulder for a long time and who protect that shoulder by elevation.

SUMMARY

1. Skeleton studies were done to show the effect on the reconstructed neurovascular tree of movements which made the tree taut and which kinked the tree in relation to muscles.

2. Personal symptoms were analysed in relation to the knowledge gained from anatomical and skeleton studies.

3. Historical developments in the diagnosis of cases of acroparaesthesia are presented.

4. Case summaries and discussions illustrate the problems involved.

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CONGENITAL AGAMMAGLOBULINAEMIA

A CASE REPORT

W. S. WINSHIP, M.B., CH.B. (CAPE TOWN), *Durban*

Agammaglobulinaemia or hypogammaglobulinaemia has been reported frequently since Bruton,[1] in 1952, first described the absence of a gamma-globulin peak in the electrophoretic pattern of a child suffering from severe recurrent infections.

It is now apparent that agammaglobulinaemia is but one effect of some still unknown primary defect, of which the absence of plasma cells from bone marrow, lymphoid tissues and bowel, and the inability to accept homografts, are other features. Deficiency of some beta-globulins has also been found in this condition, which shows itself as an inability to combat bacterial infections. Moreover, it is evident that several different forms of this deficiency occur and these have been the subject of recent reviews.[2-5]

Controversy has arisen over the use of the term agammaglobulinaemia, since the development of more sensitive methods[6,7] of determining levels of circulating gamma-globulin has shown that, in the majority of cases in which gamma-globulin is undetectable on the electrophoretic pattern, a small amount is, in fact, present. In an attempt to clarify the position, Firkin and Blackburn[4] defined agammaglobulinaemia as that condition in which gamma-globulin is not demonstrable on the electrophoretic pattern, thus perpetuating Bruton's original description, while recognizing that in many such cases very small amounts may be present. Good *et al.*[5] supported this definition as did Garvie and Kendall,[6] who considered that no change in terminology should be made until the nature of the primary defect is known.

The following is a classification of the types of agammaglobulinaemia which have been described:

1. *Primary agammaglobulinaemia* with normal total serum protein:
   - (*i*) Congenital sex-linked recessive form occurring in males.
   - (*ii*) Congenital form, so far only reported in females.
   - (*iii*) Acquired form occurring after early childhood or in adult life.
   - (*iv*) Transient self-limiting agammaglobulinaemia of infancy, occurring as an extension of the decline

in the gamma-globulin level normally found in infants between the second and sixth months of life.

II. *Secondary agammaglobulinaemia*:

(*i*) Associated with hypoproteinaemia, as found in the nephrotic syndrome.

(*ii*) Associated with neoplastic conditions, such as multiple myelomatosis and leukaemia.

It is the purpose of this paper to report a case of congenital agammaglobulinaemia, which is possibly of the sex-linked recessive form.

CASE HISTORY

C.R., a European male, now aged 3 years and 5 months (June 1961), was delivered at term by Caesarean section for disproportion. His mother was a primiparous woman aged 37 years. His birth weight was 6 lb. and his neonatal period of life uneventful. The pregnancy had been complicated by hyperemesis, and on 4 occasions between the 12th and the 24th weeks by transient vaginal bleeding. There was no known exposure to viral infections or radiation. He is the only child of unrelated parents.

At the age of 4 months he was vaccinated against smallpox and had a severe local reaction accompanied by high fever, but otherwise uncomplicated.

From the age of 6 months he suffered from repeated respiratory and enteral infections. At 15 months he became ill with diarrhoea and was treated with chloramphenicol, 125 mg. 4-hourly for 8 days. The diarrhoea stopped, but he became progressively more ill, with a persistent high fever despite the use of aspirin 4-hourly. On the eighth day of this illness he was admitted to a nursing home where he was found to have a complete absence of neutrophils in a total leucocyte count of 5,700 per c.mm. Treatment with chloramphenicol was stopped, since it was thought to be the cause of the neutropenia, and erythromycin was substituted. He remained critically ill for a further 3 days, during which time he developed several paronychiae of the fingers, a perianal abscess and bleeding from his gums. The temperature exceeded 106° F. on several occasions. He recovered after another 10 days, when his leucocytes totalled 15,150 per c.mm., of which 43% were neutrophils.

He first attended the Outpatients' Department of Addington Children's Hospital at the age of 17 months, since when he had the following illnesses:

22 May 1959 — upper-respiratory-tract infection and gingivitis.

14 July 1959 — upper-respiratory-tract infection and left otitis media, which failed to respond to treatment with penicillin, but responded to oxytetracycline after 8 days of treatment.

30 July 1959 — gastro-enteritis, which responded to treatment with neomycin and succinylsulphathiazole.

25 August 1959 — upper-respiratory-tract infection, which did not respond well to treatment with dosulphin.

On 2 October 1959, at the age of 1 year and 10 months, he was admitted to the Addington Children's Hospital with another upper-respiratory-tract infection, because he was again found to have a complete absence of neutrophils.

Examination

Physical examination in October 1959 revealed a boy 33 inches tall and weighing 27 lb. He was flushed and had a thick yellow nasal discharge. His throat was inflamed, but the tonsils were small, and there was no lymphadenopathy. His liver edge was palpated 2 inches below the costal margin, but it was soft and non-tender.

His temperature was 101·6° F., pulse rate 120 per minute and respiratory rate 24 per minute.

Laboratory Investigations

The haemoglobin was 13 G. per 100 ml., leucocytes totalled 13,000 per c.mm. (neutrophils 0%, lymphocytes 83%, monocytes 15%, eosinophils 2%), and there were 145,000 platelets per c.mm.

The bone marrow was markedly hypoplastic. Erythropoiesis was normoblastic, but depressed. All stages of myelopoiesis were present, but there was a generalized reduction in the whole series. Megakaryocytes and platelets were diminished.

The liver-function tests were normal. Zinc turbidity was 1 unit, and the serum proteins totalled 5·7 G. per 100 ml. (albumin 4·2, globulin 1·5; A:G ratio 2·8:1).

The electrophoretic pattern showed a complete absence of gamma-globulin. Descending starch-gel electrophoresis revealed a decrease in the beta-globulins as well.

His blood group was O Rhesus positive. No isoagglutinins were detected.

Family History

There is no relevant history on the paternal side, but several deaths in infancy have occurred on the maternal side, as shown in the genealogical table (Fig. 1).

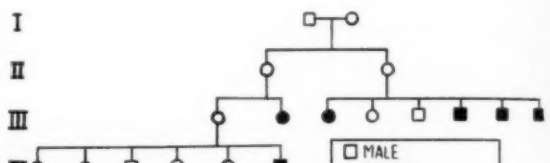

Fig. 1. Genealogical table of patients' maternal family over 5 generations (I - V).

The patient's great-grandmother had only one sibling, a sister who had 6 children. Of these, a girl died at 9 weeks, and a boy at 7 years after prolonged illness following 'infantile paralysis'. The death of the girl at 9 weeks cannot be attributed to agammaglobulinaemia, nor is it likely that agammaglobulinaemia was a factor in this boy. Two other boys, however, died at the ages of 18 months and 2 years of pneumonia, and fever with convulsions, respectively.

In the direct line, his grandmother had one sister who died at the age of 3 months, the cause of death being unknown to us, but agammaglobulinaemia is unlikely to have been a factor here because of her age and sex. Of the 6 children born to his grandmother, the first was a stillborn girl, but a boy suffered from recurrent respiratory infections and discharging ears from the age of 8 months until his death from pneumonia at 13 months.

Of the fifth generation shown in the table all are still children. Our patient is the only one living in Durban, but it is known that all the others are well, and the only other male infant has a normal electrophoretic pattern.

Treatment

From admission the child was treated with oral penicillin, which was ineffective in controlling his infection. Repeated nasal and throat swabs revealed a variety of organisms, none of which predominated. When the result of the electrophoretic pattern became known, treatment was commenced with pooled human gamma-globulin intramuscularly. Penicillin was continued and recovery took place within a few days.

The dosage of gamma-globulin was based on the work of Martin,[4] who estimated the gamma-globulin fluid compartment to be approximately 12% of the body weight. Since plasma volume is roughly 5% of the body weight, between 55% and 60% of gamma-globulin is distributed in cells and interstitial fluid, the intravascular and extravascular compartments being in equilibrium. Janeway and Gitlin[5] have found that levels of over 100 - 150 mg. are sufficient to keep agammaglobulinaemic patients free of infection, and several studies[6,7,8] have shown the half-life of gamma-globulin to be between 30 and 35 days. Maintenance of a satisfactory level can therefore be achieved by giving an injection of 0·3 - 0·4 ml. per lb. body weight of a 16% solution of gamma-globulin at 4-weekly intervals. Apart

TABLE I. SHOWING THE MARKED VARIATION IN THE NEUTROPHIL COUNT OVER A PERIOD OF 2 YEARS

Days of observation	1	3	5	8	26	109	114	147	185	188	189	191	195
Total neutrophils per c.mm.	0	0	1,431	6,300	1,590	140	320	1,520	0	600	240	0	180
Days of observation	196	198	206	220	252	282	310	348	404	468	645	716	744
Total neutrophils per c.mm.	210	1,600	1,760	3,740	0	0	1,540	720	220	0	4,080	1,440	8,840

from the advantage of less frequent injections, one large dose results in a more rapid attainment of protection and longer periods of higher circulating antibodies than do smaller weekly injections.[3] Accordingly, this child was given 0·7 ml. per lb. body weight initially, and thereafter 0·35 ml. per lb. body weight at 4-weekly intervals.

In addition, a prophylactic dose of 0·5 G. of sulphadimidine daily has been given in an attempt to prevent the upper-respiratory-tract infections to which he is particularly susceptible.

Progress

The child has been observed for 18 months since the diagnosis was made and his progress has been satisfactory. His weight gain over this period has been 7 lb. and he has grown 4 inches in height.

He has required medical attention on only a few occasions. A paronychia of the right index finger and a small post-traumatic abscess of a foot both responded rapidly to the appropriate treatment. Recently, on the day after receiving an injection of gamma-globulin, he developed acute bacillary dysentery (*Shigella sonnei*). This responded well to treatment with neomycin and succinylsulphathiazole at home. Gamma-globulin given intramuscularly takes 3 days to reach its maximum concentration in the circulation, thus the onset of dysentery occurred at a time when his gamma-globulin level was probably at its lowest.

Gingivitis and bleeding from his gums, which had been observed before admission to hospital, recurred on 3 occasions. No other evidence of a bleeding tendency was found, and his platelet count was normal. Treatment with vitamin C produced rapid improvement, and discontinuation resulted in recurrence of the bleeding. A prolonged course of vitamin C was given with an excellent outcome. His dietary intake of vitamin C at that time was very poor, whereas his intake of sweets was excessive.

During his stay in hospital, and whenever possible since his discharge, leucocyte counts, with particular reference to the neutrophils, have been made. These as well as counts done before his admission to hospital are presented in Table I. Although the number of recordings is small and over varying intervals of time, he appears to have long periods of profound neutropenia alternating with periods in which the neutrophils approach the normal number. His liver-function tests have been repeated on 4 occasions. On each occasion zinc turbidity has been 1 unit, and all other tests normal.

DISCUSSION

Cases of the congenital sex-linked recessive form of agammaglobulinaemia have been reported more often than the other forms, and a distinct clinical syndrome is apparent. It occurs in males and appears to be transmitted by females as a recessive trait. The lymphoid tissues are poorly developed in them, and absence or gross deficiency of plasma cells in the bone marrow, lymphoid tissues and bowel is a feature of these patients. There are frequently associated haematological abnormalities, the most common of which is neutropenia.[2,10,11] The neutropenia may be aregenerative or occur as cyclical or transient episodes, and is considered to be a reflection of some underlying disturbance of haemopoietic function associated with agammaglobulinaemia.[5] Lymphopenia, thrombocytopenia, and complete absence of eosinophils have also been reported.

A second type of congenital hypogammaglobulinaemia has been described affecting females.[5,12] These patients present with the same inability to combat infection. Their gamma-globulin levels, when estimated by the more sensitive immunochemical[6] or antiglobin-inhibition[7] techniques, are slightly higher than those found in the sex-linked form. There is enlargement of the lymphoid tissue including the spleen, and associated haemolytic anaemia has been described. No familial incidence has been discovered in the few cases reported so far.

In the case presented here several features suggest that the patient may have the congenital sex-linked form of agammaglobulinaemia. He is a male, his lymphoid tissues are poorly developed, and he has periodic episodes of neutropenia. Unfortunately, plasma cells were not sought for when his bone marrow was examined, and it has not been possible to repeat this examination.

It is of interest that when neutropenia was first discovered in this child it was thought to have been caused by chloramphenicol. The possibility of repeated infections or the repeated use of antibiotics causing neutropenia has been suggested, but Good and Zak[2] described a patient with agammaglobulinaemia with neutropenia, studied from birth, in which no antibiotics had been used.

Some patients with agammaglobulinaemia have succumbed to homologous serum jaundice, to which they seem particularly susceptible. Good *et al.*[5] advocated that all syringes and needles used on these patients should be of the disposable variety.

SUMMARY

A case of congenital agammaglobulinaemia, possibly of the sex-linked recessive form, is reported.

Associated periodic neutropenia is a feature of this case.

Regular monthly treatment with intramuscular injections of pooled human gamma-globulin has been effective in averting serious illness.

I am indebted to Dr. J. V. Tanchel, Medical Superintendent of Addington Hospital, for permission to publish; to Dr. F. Walt, Consultant Paediatrician, whose patient this was, for his guidance and encouragement; to Prof. H. L. Wallace for his interest and critical supervision; to Dr. B. G. Grobbelaar of the Natal Blood Transfusion Service for his helpful suggestions; and to Dr. A. Gordon of Benoni for information about a cousin of the patient.

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THE METABOLISM OF GLYCINE IN EXPERIMENTAL PORPHYRIA*†

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Ockner and Schmid[1] have recently reported a syndrome of porphyria in rats, which can be induced by feeding a diet containing 0·2% hexachlorobenzene. This syndrome has a strong biochemical resemblance to the syndrome of acute porphyria in man, and provides a convenient experimental preparation for the study of disordered porphyrin synthesis.

We have induced porphyria in this way in adult male white rats and studied the metabolism of C^{14}-labelled glycine by liver homogenates incubated *in vitro*. The liver was incubated in a standard Warburg apparatus in the presence of either C-1 or C-2 labelled glycine, and the respiratory CO_2 trapped on filter paper which had been spotted with saturated KOH. The filter paper with the absorbed radioactivity was then counted in a liquid scintillation counter. In this way the rate of conversion of each of the 2 carbon atoms of glycine to CO_2 could be compared in normal and porphyric rats.

The liver homogenates from the hexachlorobenzene-fed rats showed the following changes when compared with the normal animals (the results given are the means fo. 7 experiments):

1. A depression in Q_{O_2} (normal 9·57, porphyric 6·74).
2. A slower rate of conversion of glycine-1-carbon to CO_2 (normal 22·73 μA/G. dry weight of liver per 2 hours, porphyric 10·79 μA/G. dry weight of liver per 2 hours).
3. A slower rate of conversion of glycine-2-carbon t CO_2 (normal 6·63μA/G. dry weight of liver per 2 hours, porphyric 1·91 μA/G. dry weight of liver per 2 hours).

The depression of the rate of conversion of the second carbon atom to CO_2 was much greater than that of the first carbon atom, with the result that the ratio $\frac{C\text{-}1 \rightarrow CO}{C\text{-}2 \rightarrow CO}$ was increased in the porphyric rats (normal 3·76, porphyric 5·74).

This suggests that in hexachlorobenzene-induced porphyria there is a specific defect in the oxidation of the α-carbon atom of glycine to CO_2. It is known that the α-carbon atom of glycine can form 'active C_1 fragments', and subsequent studies with C^{14}-labelled formate have indicated that this oxidative defect might extend to all 'C fragments'.

* This work was supported by a grant P.H.S. A-3997 from the National Institute of Health, Public Health Service, USA.
† Abstract of a paper presented at Research Forum, University of Cape Town, 14 September 1961.

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IN DIE VERBYGAAN : PASSING EVENTS

Correction. An error inadvertently appeared in the article 'An analysis of the micro-organisms responsible for pelvic infection in Pretoria', which appeared in the *Journal* of 4 November 1961. In Table III, on p. 906, the figures in the *upper* half of the table refer to 'Alpha-haemolytic streptococci' and those in the *lower* half to 'Beta-haemolytic streptococci'.

* * *

Non-European Home for the Aged. Under the auspices of the Moravian Church S.A.W., the Zinzendorf Créche have started a scheme to build a home for non-European aged at Elim in the district of Bredasdorp. Obtaining the grants necessary to put this scheme into operation would be made possible only if sufficient applications are received for rooms in such a home. The names and addresses of would-be occupants must be sent to the Rev. K. Bonk, 18 Ashley Street, Cape Town, telephone 41-1918.

* * *

Dr. J. H. S. Gear, Director of the South African Institute for Medical Research, left South Africa on 4 November 1961 to attend a conference in Washington on the use of measles vaccine. Dr. Gear will spend some time at the National Institutes of Health, Bethesda, after the conclusion of the conference.

* * *

Research Forum, University of Cape Town. There will be no further meetings of Research Forum, Clinico-pathological Conferences or Clinical Conferences on Thursday afternoons until the commencement of the new academic year in 1962. Notices will be published in the *Journal* when the meetings are resumed.

* * *

Dr. M. Nellen, of Cape Town, was elected a Fellow of the Royal College of Physicians of Edinburgh at their meeting held this month.

The Natal Pathological Laboratory (Drs. Norman H. Walker and Lindsay H. Walker) will be moved to new premises in Suite 711, West Walk, West Street, Durban, as from 18 December 1961. The laboratory, which is privately owned, was opened in 1921 by Dr. N. H. Walker and has been in Chancery Buildings, Smith Street, Durban, since 1926.

* * *

South African Paediatric Association (M.A.S.A.), Natal Sub-Group. The last meeting for 1961 will be held at the Medical School, Durban, on Tuesday 5 December 1961 at 8 p.m. The meeting will take the form of a discussion on 'The physiology and correction of fluid and electrolyte loss in children'; and the opening speakers will be Prof. J. V. O. Reid and Dr. N. M. Mann.

* * *

Dr. Dudley Silove, of Johannesburg, has commenced practice as an obstetrician and gynaecologist at 520 Lister Buildings, Jeppe Street, Johannesburg. Telephones: Rooms 23-1532, residence 45-1795.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as those who change their addresses within South Africa.

* * *

Prostaphlin. With reference to the announcement which appeared in the *Journal* of 4 November (p. 922), Messrs. B. L. Pharmaceuticals have advised us that supplies of Prostaphlin will not be available in South Africa until early next year. To date only material for clinical trials has been received by them.

THE SEATS OF DISEASE

SOME NOTES TO MARK THE 200th ANNIVERSARY OF THE PUBLICATION OF MORGAGNI'S *DE SEDIBUS ET CAUSIS MORBORUM ...*

HYMIE GORDON,* B.Sc., M.D. (CAPE TOWN), M.R.C.P. (LOND.), M.R.C.P. (EDIN.)

Giovanni Battista Morgagni (1682 - 1771)

The story of Morgagni's life is not one of heroic struggle for recognition and achievement against reactionary authority and an unkind fate. He was born into a prosperous family at Forli, in Northern Italy, and when he grew up he had no difficulty in being enrolled as a medical student at the University of Bologna. He was a carefree undergraduate and, although he spent much time writing poetry and studying philosophy, he graduated without difficulty and with the highest honours. He obtained the coveted post of anatomical prosector under the great Antonio Valsalva, to whose teaching Morgagni never failed to record his debt.

After leaving Bologna, Morgagni spent a few years on postgraduate studies at the Universities of Venice and Padua before returning to Forli to open a practice in general medicine. However, the good citizens of Forli were not to benefit long from his ministrations; the authorities of Padua, whose university numbered Linacre, Galileo, Copernicus and Harvey among its more distinguished alumni, must have been impressed by the young Morgagni and, when he was only 29 years old, they invited him to become their Professor of Theoretical Medicine. Morgagni accepted their invitation and returned to Padua. Three years later, he was appointed Professor of Anatomy and thus he came to occupy the chair which a long line of illustrious predecessors — Vesalius, Fallopius, Fabricius, Gasserius and Spigelius — had already hallowed.

Unlike so many of his predecessors, Morgagni had a long and tranquil career at Padua. Unlike Galileo, he was always at peace with both Church and State and he enjoyed the friendship of several Popes and Princes. Unlike Vesalius, he had access to adequate necropsy material; in fact, it became quite fashionable in Padua to be anatomized by Morgagni, and it is recorded that several citizens requested in their wills that the great Professor should examine their mortal remains.

Giovanni Battista Morgagni

(From an original etching in the 1779 edition of *De Sedibus . . .* which is now in the medical library of the University of Cape Town.)

Morgagni published several scientific works; the one for which he is best remembered is *De Sedibus et Causis Morborum per anatomen indagatis* ('The Seats and Causes of Disease, studied anatomically'). This was published in 1761 and embodied his 50 years of clinical and pathological experience at Padua. Morgagni was 79 years old when *De Sedibus ...* appeared, but unlike Copernicus, who barely lived to see the first copies of *De Revolutionibus* off the press, he was able to spend 10 further fruitful years embellishing and producing several more editions.

Morgagni was with honour not only in his own country but throughout Europe; he was elected to honorary membership in the learned societies of Paris, Berlin and St. Petersburg, and he became a Fellow of the Royal Society of London in 1724. Apart from his medical activities, he found time to study and to contribute to the literature of history, philology and archaeology; one of his papers dealt with 'the manner of Cleopatra's death'. His personal affairs were as eminently satisfactory as his academic achievements; soon after his arrival in Padua he had married the noble lady Paola Vergieri, who provided him not only with a very handsome dowry, but also with a happy home and with a more-than-average quiverful of 15 children. And in 1771 he died as he had lived, peacefully.

De Sedibus et Causis Morborum ...

Medical journals did not exist in the early 18th century and scholars were accustomed to announce their discoveries and to propagate their ideas by way of personal communications to their colleagues. Thus, Morgagni recorded many of his clinical and pathological experiences in a series of letters to an unknown friend. Altogether, 70 such letters were written, containing the observations made at 840 necropsies. These letters were then returned to Morgagni and, after careful revision, he collated them into the 5 books which constitute the *De Sedibus ...*

The special feature of *De Sedibus ...* is the care with which the clinical manifestations are recorded and then painstakingly correlated with the findings at necropsy. Throughout the book, diseases are considered in anatomical terms, and it is for introducing this way of thinking that Morgagni has achieved such an exalted place in the history of medicine. Pathological processes are shown to produce abnormal changes in organs; in these abnormal organs are the 'seats' of the diseases which the clinician sees.

Morgagni's style tends to be long-winded and he is often pedantic. Nevertheless, the conditions which he discusses are usually quite easily recognized and the modern reader is delighted by the many intimate glimpses which his clinical notes provide of the persons and of the social and medical practices of the time. For example, the ninth letter includes the case history of:

'my fellow-citizen Anastasio Poggi, a grave and worthy priest. He was in his sixty-eighth year, of a habit moderately fat, and of a florid complexion,

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when he was first seiz'd with the epilepsy, which left behind it the greatest slowness of the pulse and in like manner a coldness of the body.'*

Father Anastasio was offered a variety of remedies, from vermifuges to venesection, and eventually it was found that half a grain of opium taken at night prevented the attacks. When the opium was stopped, the attacks returned.

'And, indeed, after he had pass'd a night of that kind, which was far more troublesome than the rest, when to the greatest rarity of the pulse . . . an inequality had suddenly been added, so that very often they were perceiv'd to be even much more rare, then not more so than usual, and presently much rarer again; which symptom made us the more uneasy, because the disease, at that time, was wont, first of all, entirely to obscure the pulse, and then immediately to begin its attack; and when we had tried all the remedies, recommended to dissolve and promote, the circulation of the blood, in vain; upon giving the opium again, the quiet nights again return'd, and diminish'd that inequality of the pulse: and, by continu'd use of opium every night, it was entirely remov'd, and even the former rarity was diminish'd.'

This seems to have been a case of heart block with a variable ventricular response and occasional periods of ventricular asystole; this syndrome is now associated eponymously with the Irish physicians, Adams and Stokes who, in 1827 and 1846 respectively, described similar cases. But Morgagni is not lacking in eponymous fame; the 20th edition of Dorland's *Illustrated Medical Dictionary* lists no fewer than 25 entities with which his name is associated: there are caruncles, cataracts, columns and crypts of Morgagni; 'Morgagni's disease' is hyperostosis frontalis; 'Morgagni's liquor' is the fluid between the eye lens and its capsule; and the 'hydatid of Morgagni' is the cyst-like remnant of the Müllerian duct attached to the oviduct or testis!

Fine descriptions of cirrhotic livers, renal calculi and typhoid ulcers all testify to Morgagni's ability as a special pathologist. He deals with most of the common neoplasms, but he does not appear to have understood the significance of metastases. He describes very strikingly the clinical features in the case of a Paduan nun with lobar pneumonia and his comparison of her consolidated lungs at necropsy with liver tissue introduced 'hepatization' into the jargon of the morbid anatomist. The sections on consumption are poor; this may be because of Morgagni's fastidious reluctance to handle contagious material.

Morgagni practised medicine in the post-Harveian era, when the doctrine of the circulation of the blood was firmly established. It is not surprising, therefore, that the best chapters of his book are those which deal with the circulatory system. Cerebral vascular lesions are described in profusion. There are good accounts of mitral and aortic stenosis and of atherosclerosis of the aorta and coronary vessels. Aortic aneurysms are well described and their syphilitic origin is mooted. The following extract from *De Sedibus* . . . deals with a case of aortic aneurysm; it is quoted at length to illustrate Morgagni's combined clinical-pathological approach and because it provides an enchanting vignette of 18th century medical practice.

Letter the Twenty-sixth

Treats of sudden death, from a disorder of the sanguiferous vessels, especially those that lie in the thorax.

A man who had been too much given to the exercise of tennis and the abuse of wine, was, in consequence of both these irregularities, seiz'd with a pain of the right arm, and soon after of the left, join'd with a fever. After these there appear'd a tumour on the upper part of the sternum, like a large boil: by which appearance some vulgar surgeons being deceiv'd, and either not having at all observ'd, or having neglected, the pulsation, applied such things as are generally us'd to bring these tumours to suppuration; and these applications were of the most violent kinds. As the tumour still encreas'd, others applied emollient medicines, from which it seem'd to them to be diminish'd; that is, from the fibres being rubb'd with ointments and relax'd; whereas they had been before greatly irritated by the applications. But as this circumstance related rather to the common integuments, than to the tumour itself, or to the coats that were proper thereto, it not only soon recover'd its former magnitude, but even was, plainly seen to encrease every day. Wherefore, when the patient came into the Hospital of Incurables, at Bologna, which was, I suppose, in the year 1704, it was equal in size to a quince; and what was much worse, it began to exsude blood in one place: so that the man himself was very near having broken through the skin (this being reduc'd to the utmost thinness in that part, and he being quite ignorant of the danger which was at hand) when he began to pull off the bandages, for the sake of showing his disorder. But this circumstance being observ'd, he was prevented going on, and order'd to keep himself still, and to think seriously and piously of his departure from this mortal life, which was very near at hand, and inevitable. And this really happen'd on the day following, from the vast profusion of blood that had been foretold, though not so soon expected by the patient. Nevertheless, he had the presence of mind, immediately as he felt the blood gushing forth, not only to commend himself to God, but to take up with his own hands a bason that lay at his bed-side; and, as if he had been receiving the blood of another person, put it beneath the gaping tumour, while the attendants immediately ran to him as fast as possible, in whose arms he soon after expired.

In examining the body before I dissected it, I saw that there was no longer any tumour, inasmuch as it had subsided after the blood, by which it had been rais'd up externally, had been discharg'd. The skin was there broken through, and the parts that lie beneath it with an aperture, which admitted two fingers at once. The membrana adiposa of the thorax discharged a water during the time of dissection, with which some vessels were also turgid, that were prominent, here and there, upon the surface of the skin in the feet and the legs. In both the cavities of the thorax, also, was a great quantity of water, of a yellowish colour. And there was a large aneurism, into which the anterior part of the curvature of the aorta itself being expanded, had partly consum'd the upper part of the sternum, the extremities of the clavicles, which lie upon it and the neighboring ribs, and partly had made them diseas'd, by bringing on a caries. And where the bones had been consum'd or affected with the caries, there not the least traces of the coats of the artery remain'd: to which, in other places, a thick substance everywhere adher'd internally, resembling a dry and lurid kind of flesh, distinguish'd with some whitish points: and this substance you might easily divide into many membranes, as it were, one lying upon another, quite different in their nature from those coats to which they adher'd, as they were evidently polypous. And these things being accurately attended to, nothing occur'd besides that was worthy of remark.

* This and subsequent quotations are taken from the English translation by Benjamin Alexander, published in London in 1769.

Morgagni's Place in the History of Pathology

Before Morgagni, only faint glimmerings of the science of pathology can be discerned. The physician Antonio Benivieni, who practised in Florence during the height of the Renaissance, was the first person to conduct necropsies with the deliberate intention of determining the causes of obscure illnesses. His clinical-pathological case notes were published posthumously under the title of *De Abditis Nonullis ac Mirandis Morborum et Sanationum Causis* ('Concerning some hidden and remarkable causes of diseases and cures'; 1507). During the latter half of the next century, Théophile Bonet, a graduate of Bologna, collected together some 3,000 clinical-pathological case reports, representing all that had been written on this subject since ancient times. In 1679, these were published in a 3-volume encyclopaedia entitled *Sepulchretum sive Anatomia Practica ex Cadaveribus Morbo Denatis*. This book made no new contributions to knowledge, but much which had been forgotten was brought back to light. It was a popular and influential reference book for almost a century.

Morgagni, however, was dissatisfied with *Sepulchretum;* the information was poorly organized, many of the reports were inadequate, and Bonet was entirely uncritical in his selection of material; above all, no attempt was made to explain the individual clinical manifestations in terms of the findings *postmortem*. In *De Sedibus* . . . Morgagni set out to remedy these defects and, considering the technical limitations of the time, he was remarkably successful. Only macroscopic examination was possible and Morgagni was unable to track the seats of diseases further than to the affected organs, but this in itself was a tremendous achievement. His contribution, therefore, was to establish special pathology on a secure basis; he did not advance the knowledge of general pathology and he adhered conventionally to the humoral theories of the ancients. Further technical progress was required before the science of general pathology could be extended.

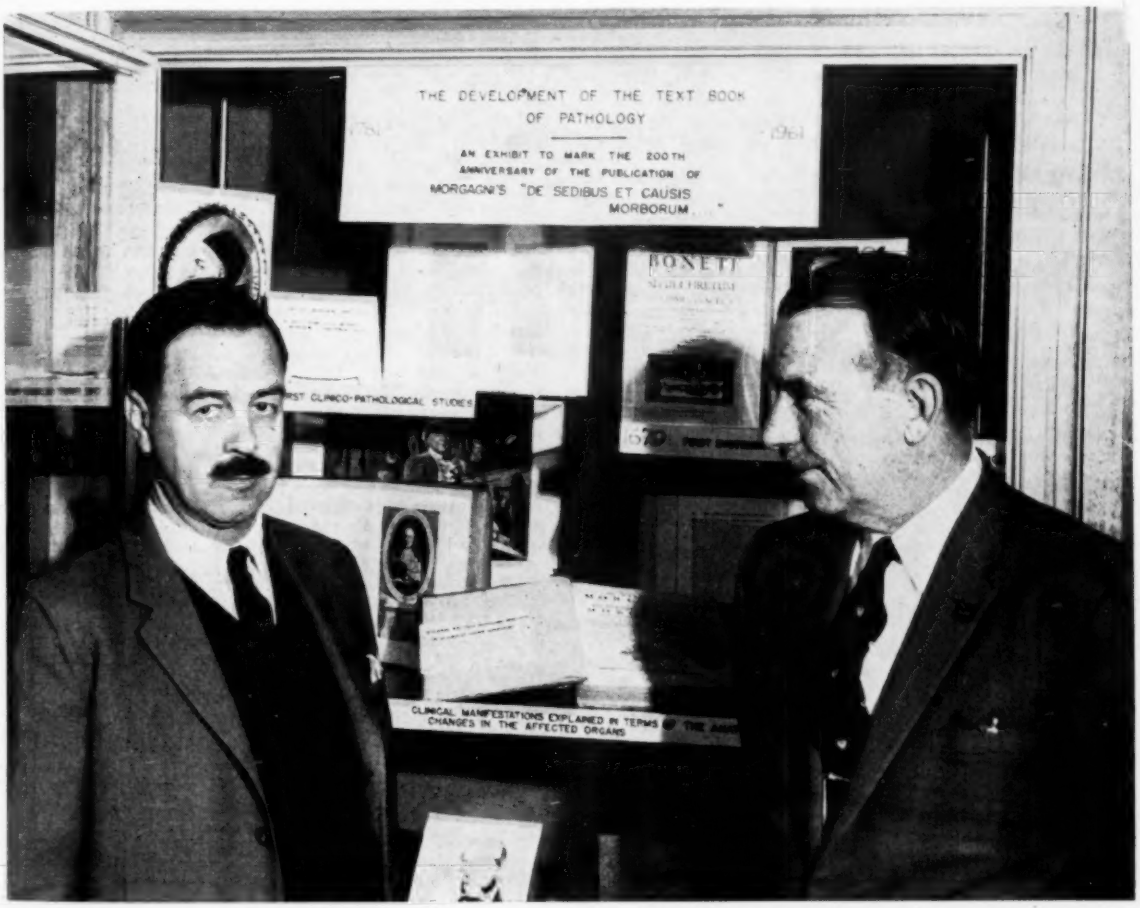
This necessary stimulus to progress came when finer dissection techniques, and particularly microscopy, began to be used extensively. Marie-François-Xavier Bichat of Paris made the most of the new techniques. He was chiefly responsible for introducing histology into the pathological armamentarium. In 1801 and 1802, he published a vast *Anatomie Generale* in which he traced the seats of disease beyond the organs into the various tissues of the body. Thus disease processes were considered to arise in nervous tissue, muscular tissue, osseous tissue, connective tissue, and so on.

With greater experience and refinement in microscopic techniques, further advances were inevitable and the great Rudolf Virchow is credited with being chiefly responsible for showing that it is not in the tissues, but in the cells from which the tissues are made that the seats of diseases are to be found. He demonstrated that cells did not develop out of body fluids but were derived from other cells (*'omnis cellula e cellula'*). Diseased cells are those which arise from the action of irritative influences on normal cells; depending on the nature of the irritant and on the cell's response, irritation could lead to the death of the cell, or to an inflammatory change, or to its benign or malignant proliferation. These concepts were

elaborated by Virchow in the greatest of his many works: *Die Cellularpathologie in ihrer Begründung auf physiologische und pathologische Geweblehre* ('Cellular pathology: its foundation in physiological and pathological histology'; 1858).

Virchow was an enthusiastic student of general and medical history and his study of the development of the ideas of pathology enabled him to foresee that further progress would come not from histological, but from biochemical, research. In this he has been proved to be quite correct; today we recognize the seats of diseases to be in the sub-microscopic molecular units of the cells — in the inborn or acquired abnormalities of specific amino acids, polysaccharides, nucleic acids and so forth. The stream of progress is still flowing strongly and some students are beginning to look beyond the molecules to the ultimate energy constituents of matter and of life.

But beyond that, dare one look further?



Dr. W. A. D. Anderson, of Miami, Florida, USA, guest professor of pathology at the University of Cape Town, opening an exhibition illustrating the development of textbooks of pathology to mark the 200th anniversary of the publication of Morgagni's *De Sedibus et Causis Morborum* . . . With Professor Anderson, is Prof. B. Bromilow-Downing, Dean of the Faculty of Medicine, University of Cape Town. The exhibition, which was opened on 22 September 1961, was at the Medical Library of the University of Cape Town, and was organized by the University of Cape Town Medical History Club. It included original editions, facsimiles, photographs and morbid anatomical specimens, to illustrate the works of Benivieni, Bonet, Morgagni, Rokitansky, Virchow, Aschoff, Muir and Anderson. The 1961 edition of Professor Anderson's *Textbook of Pathology* arrived just in time to be included in the exhibition.

AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENT

MEDIESE HULPVERENIGINGS

Lede van die Vereniging word hiermee in kennis gestel dat die reglemente van die Mediese Hulpvereniging van die Poskantoor verander is te dien effekte dat die hulpvereniging nie verantwoordelik sal wees vir die betaling van medisyne wat na 30 November 1961 vir lede van die hulpvereniging of hul afhanklikes berei of aan hulle verskaf word nie.

MEDICAL AID SOCIETIES

Members of the Association are advised that the Post Office Medical Aid Society has amended its rules to the effect that the society will not be responsible for the payment of medicines dispensed or supplied to members of the society or their dependants after 30 November 1961.

Die Natal Industries Medical Aid Society het kennis gegee dat eisvorms met ingang 1 Januarie 1962 afgeskaf gaan word. Die enigste vereiste sal wees dat lede van die hulpvereniging die gespesifiseerde rekenings wat deur geneeshere aan hulle gelewer word, moet onderteken.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
8 November 1961

The Natal Industries Medical Aid Society has advised that claim forms will be abolished as from 1 January 1962. The only requirement will be that members of the society will have to sign the specified accounts rendered to them by their medical practitioners.

L. M. Marchand
Associate Secretary

28 Plaza Buildings
Pretoria
8 November 1961

BRIEWERUBRIEK : CORRESPONDENCE

EXAMINATION FOR THE FELLOWSHIP OF THE FACULTY OF RADIOLOGY OF LONDON

To the Editor: Notices in your *Journal* in previous years that an examination for the F.F.R. could be arranged in South Africa if there were sufficient candidates, did not result in any radiologists signifying that they would sit for such an examination.

As a Congress of Radiologists is being held in Rhodesia in May of next year and as it is understood that several prominent radiologists may be coming from England to this Congress, this would facilitate the holding of an examination for the F.F.R. in Johannesburg. The expense involved in bringing out examiners would be greatly reduced.

Would any radiologists who wish to take the examination, either in Therapy or Diagnosis, for the F.F.R. communicate with me.

The South African College of Physicians and Surgeons will not be able to arrange examinations for the Fellowship in Radiology in 1962, although it is hoped that arrangements may be finalized for F.F.R. examinations by 1963.

M. Weinbren
Chairman of the Committee Representing the Faculty of Radiology in London

X-Ray Department
Chamber of Mines Hospital
P.O. Box 774
Johannesburg
8 November 1961

THE LANGUAGE OF THE PATIENT

To the Editor: 'You can be proud, when I say that there are few people so little conscious of race in this country, as are the members of the Medical Profession'—Alan Paton. (University of Natal Medical Graduates Dinner, October 1961.)

Had I been in the position of the distinguished speaker, I would have said: You should be ashamed that, as the profession least conscious of race in this country, so few of you working among non-European patients have made the slightest attempt to learn the language of the patient, or a language mutually known to yourselves and the patients.

Of course, the whole question is: 'Is it really necessary for the doctor to be able to talk the language of his patient? Will not an interpreter be just as good?' This question can only be answered by the very small percentage of doctors who have had to struggle with the use of interpreters before being able to speak to their patients directly. It is then that one realizes that by using an 'occasional' interpreter — the commonest method of obtaining a history — only about 50% accuracy can be achieved. The sooner this is realized, the more reluctant, one hopes, will doctors be to publish 'information' that they have obtained second-hand (and a poor second-hand) from these interpreters.

It is with considerable interest that one saw recently in the popular press that about 4,000 doctors in America were struck off the register because they could not speak English with the proficiency that was deemed desirable for their being good doctors. Knowing the profession in that 'cradle of apartheid', one wonders exactly what the number would have been had the doctors in 'The land of the free' needed to learn an African tongue to remain on the register! It is most reassuring to see that the Americans are prepared to act positively in regard to such people. One could imagine the look of scorn on the face of a medical superintendent in Britain, were an African doctor to apply for a post as houseman without being proficient in the English language. Why then, should our African patients have to submit to the services of doctors who are not prepared to try to establish the direct doctor-patient relationship that is especially important in the handling of illiterate and poor patients?

Perhaps the most extraordinary thing is that this attitude would appear to be fully condoned by those in charge of medical services in this country. I believe that I am right in saying that on no single occasion have directives ever been issued by medical directors in any of the four Provinces, saying that it is obligatory (or even desirable) for doctors to be able to speak to their patients directly. Furthermore, the fact that there are doctors who have worked in hospitals among African patients in a full-time capacity for 10 and often over 20 years, without having made even the slightest effort to learn the patient's language, would indicate to anyone an intellectual attitude towards the art of healing that is slovenly in the extreme. Doctors in this country are by no means alone in this matter: during a recent visit to Southern Rhodesia I was appalled by an attitude that appeared even more negative and distressing than that prevalent among the profession here.

We live at a time when the poorer non-European patient is beginning to realize the benefits of a 'proper' doctor. One cannot help hoping that the days of the interpreter-using, calcium gluconate-injecting witch-doctor (and indeed there are certain registered doctors who are little better), are numbered. The overwhelming majority of urban Africans now expect to be treated as civilized patients when they come to hospital for treatment: they expect to have a decent history taken (unlike some of their country cousins, perhaps) and are grateful for being thoroughly examined. They are particularly anxious to speak directly to the examining doctor, but this is a privilege seldom granted them, since in about 90% of consultations, 'contact' is mediated *via* an interpreter.

I should like to end by saying that if it is unethical for any doctor to publish information that he does not personally know, or personally believe, to be correct, then it is unethical to publish 'information' obtained second-hand through an interpreter. It is my sincere hope that in this country those doctors who are unable to speak the patient's own language or a language mutually understood by patient and doctor, should now make an attempt to learn such a language, especially those contemplating writing clinical papers. People possessing the intellect that is commonly attributed to members of the medical profession will have little difficulty in doing this if they are prepared to make even the slightest attempt.*

G. D. Campbell

1117 Colonial Mutual Buildings
West Street
Durban
8 November 1961

[* See also the Editorial article which is published on p. 985 of this issue of the *Journal* — Editor.]